

The validity of classification for the clinical presentation of intracranial dural arteriovenous fistulas

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✓ A number of classification schemes for intracranial dural arteriovenous fistulas (AVFs) have been published that claim to predict which lesions will present in a benign or aggressive fashion based on radiological anatomy. We have tested the validity of two proposed classification schemes for the first time in a large single-institution study.

A series of 102 intracranial dural AVFs in 98 patients assessed at a single institution was analyzed. All patients were classified according to two grading scales: the more descriptive schema of Cognard, *et al.* (Cognard) and that recently proposed by Borden, *et al.* (Borden). According to the Borden classification, 55 patients were Type I, 18 Type II, and 29 Type III. Using the Cognard classification, 40 patients were Type I, 15 Type IIA, eight Type IIB, 10 Type IIA+B, 13 Type III, 12 Type IV, and four Type V.

Intracranial hemorrhage (ICH) or nonhemorrhagic neurological deficit was considered an aggressive presenting clinical feature. A total of 16 (16%) of 102 intracranial dural AVFs presented with hemorrhage. Eleven of these hemorrhages (69%) occurred in either anterior cranial fossa or tentorial lesions. When analyzed according to the Borden classification, none (0%) of 55 Type I intracranial dural AVFs, two (11%) of 18 Type II, and 14 (48%) of 29 Type III intracranial dural AVFs presented with hemorrhage ($p < 0.0001$). After exclusion of visual or cranial nerve deficits that were clearly related to cavernous sinus intracranial dural AVFs, nonhemorrhagic neurological deficits were a feature of presentation in one (2%) of 55 Type I, five (28%) of 18 Type II, and nine (31%) of 29 Type III patients ($p < 0.0001$). When combined, an aggressive clinical presentation (ICH or nonhemorrhagic neurological deficit) was seen most commonly in intracranial dural AVFs located in the tentorium (11 (79%) of 14) and the anterior cranial fossa (three (75%) of four), but this simply reflected the number of higher grade lesions in these locations. Aggressive clinical presentation strongly correlated with Borden types: one (2%) of 55 Type I, seven (39%) of 18 Type II, and 23 (79%) of 29 Type III patients ($p < 0.0001$). A similar correlation with aggressive presentation was seen with the Cognard classification: none (0%) of 40 Type I, one (7%) of 15 Type IIA, three (38%) of eight Type IIB, four (40%) of 10 Type IIA+B, nine (69%) of 13 Type III, 10 (83%) of 12 Type IV, and four (100%) of four Type V ($p < 0.0001$).

No location is immune from harboring lesions capable of an aggressive presentation. Location itself only raises the index of suspicion for dangerous venous anatomy in some intracranial dural AVFs. The configuration of venous anatomy as reflected by both the Cognard and Borden classifications strongly predicts intracranial dural AVFs that will present with ICH or nonhemorrhagic neurological deficit.

KEY WORDS • dural arteriovenous fistula • cerebral arteriovenous malformation • cerebral hemorrhage • meninges

INTRACRANIAL dural arteriovenous fistulas (AVFs) are estimated to occur only one-tenth as frequently as intraparenchymal brain arteriovenous malformations (AVMs).¹⁵ Although rare, much information has been gathered from a metaanalysis of many small personal series, case reports, and location-specific studies.² One large heterogeneous study was published recently.⁶

Early investigators realized the potential danger of these lesions but believed that all of them had the potential for aggressive behavior.^{10,16} As a basis for understanding these complex lesions, investigators have searched for factors with which they might draw some connection between the lesions' pathophysiology and clinical presentation. Cortical venous drainage and venous ectasia were

first correlated with intracranial hemorrhage (ICH) by Houser and colleagues⁹ in 1972; this was confirmed by Castaigne, *et al.*,⁵ who identified a 42% hemorrhage rate in their series. Cortical venous drainage, venous ectasia, and lesion location "outside a major sinus" were significant in the 1984 metaanalysis of Malik, *et al.*¹⁴ The concept of progressive restriction of sinus outflow was subsequently incorporated into a classification scheme for transverse sinus intracranial dural AVF by Lalwani and coworkers.¹¹ In 1986, Lasjaunias and associates¹³ demonstrated that venous drainage characteristics determined the type of symptoms that were manifested and that focal neurological deficits were a function of the territory of venous drainage. This was clearly demonstrated by Awad and col-

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leagues² review of 360 cases that had been reported in the literature prior to 1990. They found that lesion location and factors related to the venous drainage of the intracranial dural AVF (leptomeningeal venous drainage, venous ectasia, and galenic venous drainage) correlated with ICH and nonhemorrhagic neurological deficit at presentation.

The first comprehensive classification of intracranial dural AVFs based on radiological anatomy was proposed by Djindjian and colleagues⁷ in 1977; it has subsequently been modified by Cognard, *et al.*⁶ (Cognard). In their series of 205 patients, Cognard, *et al.*, were able to show a relationship between type and aggressive presentation. More recently a similar, but simplified, version of this classification was proposed by Borden, *et al.*⁴ (Borden). They used five cases to demonstrate how their classification might be applied, suggesting its efficacy as a mode of predicting lesion behavior and how it might serve as a rationale for treatment.

In the literature focusing on intracranial dural AVFs, there has been a lack of accord concerning the terms used to describe an important configuration of venous anatomy that allows blood to flow in a retrograde manner to brain structures from the dura-based nidus. Terms used by other authors include "cortical venous drainage" and "subarachnoid venous drainage;" however, we will discuss the importance of the term "retrograde leptomeningeal venous drainage" over other misleading descriptions in our assessment of intracranial dural AVFs.

Our series represents our experience with intracranial dural AVFs of all types at a single large institution. For the first time, we examine the efficacy of the Borden classification in predicting aggressive lesion behavior at presentation. The Cognard classification is evaluated for its reproducibility and for any additional correlative value over that of the Borden categories. In defining the validity of these classification schemes we aim to explain the link between venous anatomy and clinical presentation.

Clinical Material and Methods

A review of all intracranial dural AVFs seen by the University of Toronto Brain Vascular Malformation Study Group between 1984 and July 1995 was performed. Data collection was retrospective in cases prior to 1991, obtained by a review of hospital records and those of referring specialists. Data collection was prospective from 1991 onward.

A series of 102 intracranial dural AVFs in 98 patients is presented. The following clinical data were collected: age, gender, date of presentation, onset of symptoms or signs, presenting clinical features, prior treatment, and potential predisposing factors. All angiograms were independently assessed by two neuroradiologists for the following features: location of nidus, arterial supply, venous drainage, presence of retrograde leptomeningeal venous drainage or abnormal morphological features, and computerized tomography (CT) or magnetic resonance (MR) imaging findings. In addition, these neuroradiologists independently graded all intracranial dural AVFs according to both the Cognard and Borden classifications (Fig. 1 and Table 1). In addition, we have deliberately substituted the term "retrograde leptomeningeal venous drainage" for the original terms: "cortical venous drainage" used by Cognard,

TABLE 1

Summary of classification schemes used to determine intracranial dural AVFs*

Borden Classification	Cognard Classification
Type I: DVS/MV outflow only	Type I: DVS/MV outflow only (anterograde) Type IIA: DVS/MV outflow only (retrograde)
Type II: DVS/MV outflow & RLVD	Type IIB: DVS/MV outflow (anterograde) & RLVD Type IIA+B: DVS/MV outflow (retrograde) & RLVD
Type III: RLVD only	Type III: RLVD only (no ectasia) Type IV: RLVD only (ectasia) Type V: RLVD only (spinal)

* Borden classification is presented in Borden JA, Wu JK, Shucart WA: *J Neurosurg* 82:166-179, 1995; Cognard classification is presented in Cognard C, Gobin YP, Pierot L, et al: *Radiology* 194:671-680, 1995. Abbreviations: AVF = arteriovenous fistula; DVS = dural venous sinus; MV = meningeal vein; RLVD = retrograde leptomeningeal venous drainage.

et al., and "subarachnoid venous drainage" used by Borden, *et al.*

Statistical Analysis

Statistical analysis of results was made using chi-square testing of contingency tables. In cases in which the validity of the chi-square analysis is questionable because of low values, it is indicated by a double asterisk (**).

Results

A series of 102 intracranial dural AVFs in 98 patients was analyzed (four patients harbored two separate lesions). There were 53 males and 45 females in this group with a mean age of 60 years (range 2-78 years). The most common nidus locations were the transverse sinus (44%), cavernous sinus (29%), and tentorium (14%). When the Borden classification was applied, 55 intracranial dural AVFs (54%) were found to be Type I, 18 (18%) Type II, and 29 (28%) Type III. All anterior cranial fossa lesions were Type III and all tentorial lesions were either Type II or III. The majority of cavernous sinus and transverse sinus lesions were Type I. The Cognard classification resulted in the following distribution of intracranial dural AVFs: 40 Type I, 15 Type IIA, eight Type IIB, 10 Type IIA+B, 13 Type III, 12 Type IV, and four Type V. The relationship between type and location is shown in Table 2.

A clear history of potential etiological factors was identified in 31 (32%) of the 98 patients: trauma in 23 patients, surgery in four, tumor in two, and infection in two; potential causes were unknown in the other 67 patients. Two of the four surgeries consisted of gasserian ganglion glycerol injections and both of the cases associated with non-surgically treated tumors presented after a diagnosis of meningioma involving a dural venous sinus. There were two cases of severe middle ear infections presumed to have dural venous sinus involvement on that side. Benign symptoms were found to be associated with the nidus location. All patients with cavernous sinus lesions presented with headache, bruit, orbital venous hypertension,

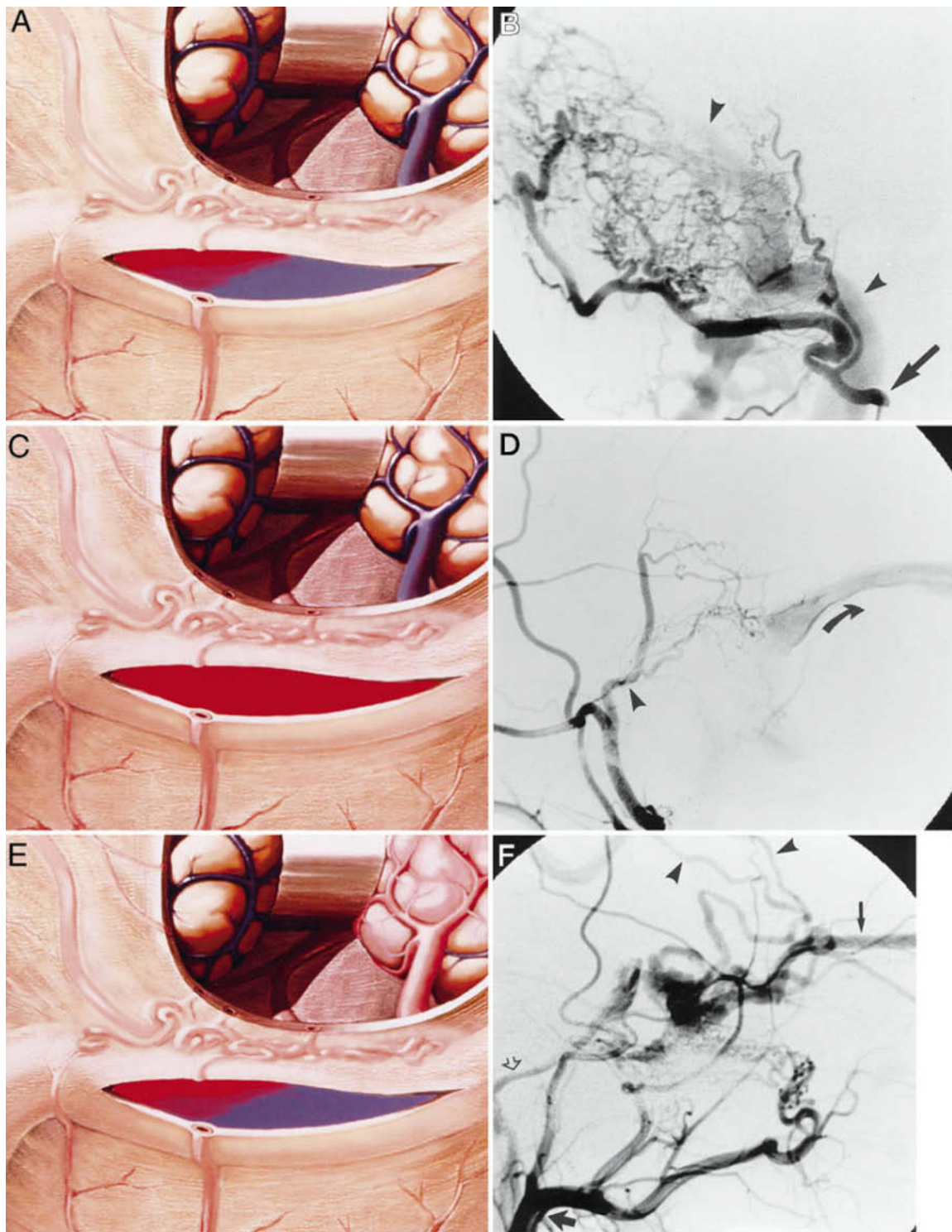
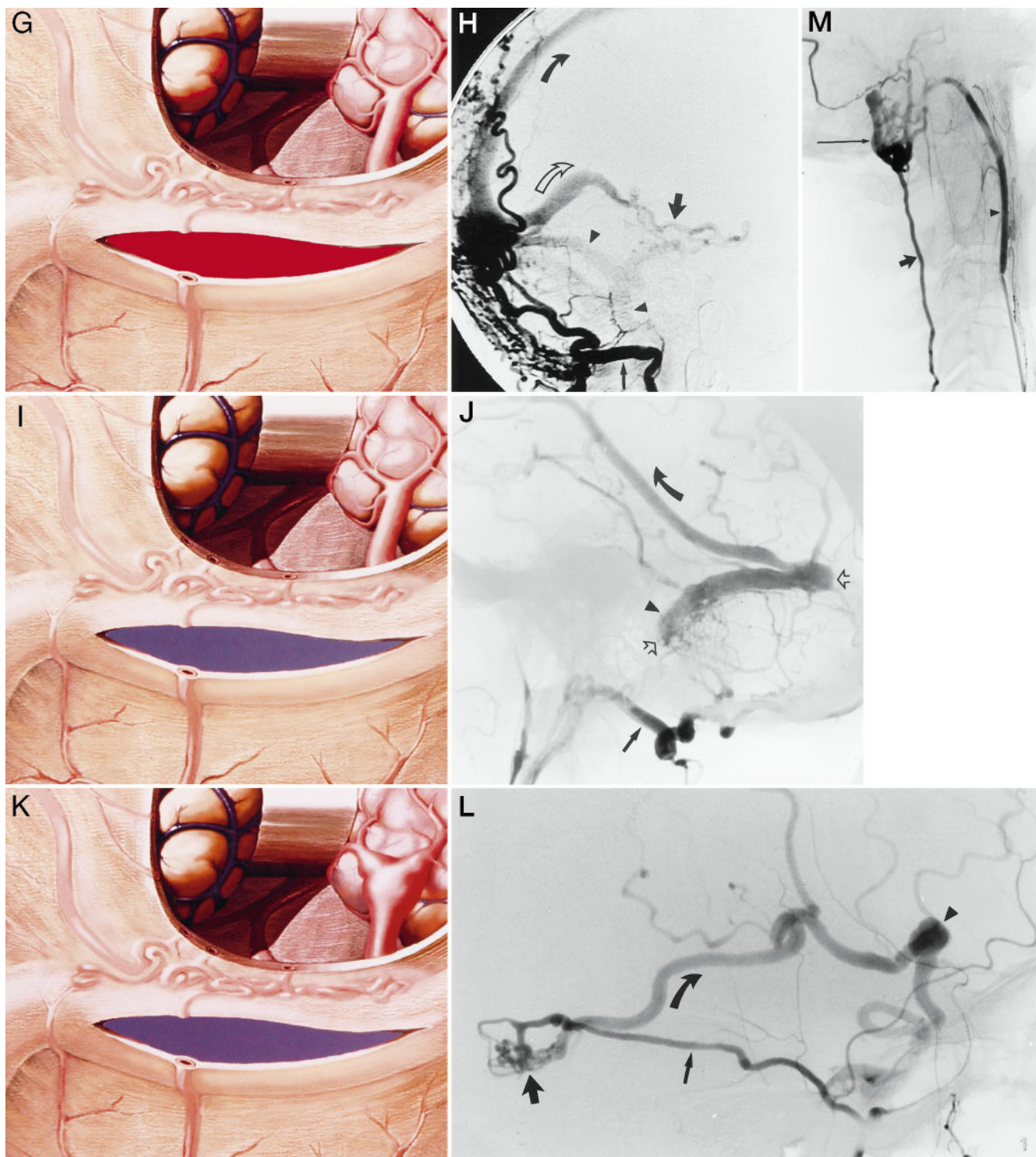


FIG. 1. Stylized artist's representations of standard left transverse sinus (TS) intracranial dural arteriovenous fistulas (AVFs) of different types compared with same type cerebral angiographic images. The artist's images illustrate the nidus in the wall of the TS, which is opened longitudinally to reveal the varying content of arterial (*red*) and venous (*blue*) blood to suggest flow patterns. Dural arteries within the convexity dura are seen coursing to supply the nidus. A window in the dura above the TS has been opened to reveal the occipital lobe being retracted to expose a feeding artery within the tentorial dura and a leptomeningeal vein coursing from the brain to the TS. A and B: Borden Type I/Cognard Type I intracranial dural AVF, which drains into meningeal vein(s) or dural venous sinus (DVS), with anterograde DVS flow. Artist's rendering (A) displaying well-oxygenated blood (*red*) exiting the nidus as it mixes with the normal anterograde flow (*blue*) in the TS. There is no retrograde leptomeningeal venous drainage. Selective right occipital artery (*arrow*) angiogram, lateral projection (B) demonstrating a diffuse TS nidus that drains anterogradely into the TS and sigmoid sinus (*arrowheads*). C and D: Borden Type I/Cognard Type IIA intracranial dural AVF draining into the meningeal vein(s) or DVS with retrograde DVS flow. (FIG. 1 *continued* →)

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Artist's rendering (C) demonstrating retrograde DVS flow (red blood exiting the nidus fills the TS). There is no retrograde leptomeningeal venous drainage. Left distal external carotid artery angiogram, lateral projection (D), showing middle meningeal (*arrowhead*) branches feeding a TS nidus. Only a retrograde flow is seen in the TS (*arrow*). E and F: Borden Type II/Cognard Type IIB intracranial dural AVF exhibiting retrograde leptomeningeal venous drainage as well as drainage into the meningeal vein(s) or DVS with anterograde DVS flow. Artist's rendering (E) displaying nidal outflow (*red*) as it mixes with the normal anterograde TS flow (*blue*). A leptomeningeal vein (*now red*) is also involved in the lesion's venous drainage (retrograde leptomeningeal venous drainage). Right external carotid artery (*large arrow*) angiogram, lateral projection (F) of a cavernous sinus intracranial dural AVF with retrograde leptomeningeal venous drainage into the cortical veins (*arrowheads*) and anterograde flow through the inferior petrosal sinus (*open arrow*). Reflux into the superior ophthalmic vein (*small arrow*) is seen. G and H: Borden Type II/Cognard Type IIA+B intracranial dural AVF displaying retrograde leptomeningeal venous drainage, but also drainage into meningeal vein(s) or DVS with retrograde DVS flow. Artist's rendering (G) demonstrating (FIG. 1 *continued*→)

TABLE 2
Types and locations of 102 intracranial dural AVFs*

Classification & Type	Location				Other
	ACF	Cavernous Sinus	Ten-torium	Transverse Sinus	
Borden Type I					
Cognard Type I	0	15	0	22	3
Cognard Type IIA	0	8	0	7	0
Borden Type II					
Cognard Type IIB	0	4	2	1	1
Cognard Type IIA+B	0	3	1	6	0
Borden Type III					
Cognard Type III	2	0	5	4	2
Cognard Type IV	2	0	4	4	2
Cognard Type V	0	0	2	1	1
total	4	30	14	45	9

* Borden classification is presented in Borden JA, Wu JK, Shucart WA: *J Neurosurg* 82:166–179, 1995; Cognard classification is presented in Cognard C, Gobin YP, Pierot L, et al: *Radiology* 194:671–680, 1995. Abbreviations: ACF = anterior cranial fossa; AVF = arteriovenous fistula; other = foramen magnum, superior sagittal sinus, or middle cranial fossa.

or cranial nerve and visual deficits. The majority of patients with transverse sinus intracranial dural AVFs experienced headache and bruit as their only benign symptom, as did patients with lesions in the middle cranial fossa and foramen magnum. Headache was the predominant benign symptom in tentorial lesions.

An aggressive clinical presentation was regarded as one leading to death, ICH, or nonhemorrhagic neurological deficit (other than local cranial nerve phenomena associated with cavernous sinus lesions). Hemorrhage was a presenting feature in 16 (16%) of 102 intracranial dural AVFs. This was a dominant feature in both anterior cranial fossa (two (50%) of four) and tentorial (nine (64%) of 14) lesions. Combining both ICH and nonhemorrhagic neurological deficit, an aggressive presentation was a feature of three anterior cranial fossa (75%), 11 tentorial (79%), three foramen magnum (60%), and 13 transverse sinus (29%) lesions. No patient had a cavernous sinus lesion that presented in an aggressive manner.

Classification by Borden type showed the following distribution of presentation with ICH: Type I, none (0%) of 55; Type II, two (11%) of 18; Type III, 14 (48%) of

TABLE 3
Relationship between aggressive clinical presentation and classification of 102 intracranial dural AVFs*

Classification & Type	Presentation		
	ICH	NHND	ICH & NHND
Borden Type I (55 AVFs)	0 (0%)	1 (2%)	1 (2%) [†]
Cognard Type I (40 AVFs)	0 (0%)	0 (0%)	0 (0%) [‡]
Cognard Type IIA (15 AVFs)	0 (0%)	1 (7%)	1 (7%)
Borden Type II (18 AVFs)	2 (11%)	5 (28%)	7 (39%)
Cognard Type IIB (8 AVFs)	1 (13%)	2 (25%)	3 (38%)
Cognard Type IIA+B (10 AVFs)	1 (10%)	3 (30%)	4 (40%)
Borden Type III (29 AVFs)	14 (48%)	9 (31%)	23 (79%)
Cognard Type III (13 AVFs)	5 (38%)	4 (31%)	9 (69%)
Cognard Type IV (12 AVFs)	6 (50%)	4 (33%)	10 (83%)
Cognard Type V (4 AVFs)	3 (75%)	1 (25%)	4 (100%)

* Borden classification is presented in Borden JA, Wu JK, Shucart WA: *J Neurosurg* 82:166–179, 1995; Cognard classification is presented in Cognard C, Gobin YP, Pierot L, et al: *Radiology* 194:671–680, 1995. Abbreviations: AVF = arteriovenous fistula; ICH = intracranial hemorrhage; NHND = nonhemorrhagic neurological deficit.

[†] $\chi^2 = 54.644$, $df = 2$, $p < 0.0001$.

[‡] $\chi^2 = 50.288$, $df = 2$, $p < 0.0001$. Because of low values, the validity of the chi-square analysis is questionable.

29; ($\chi^2 = 33.806$, $df = 2$, $p < 0.0001$). A similar correlation was found using the Cognard classification ($\chi^2 = 50.288^{**}$, $df = 2$, $p < 0.0001$). Aggressive nonhemorrhagic neurological deficits (as defined above) were an element of presentation in 15 (15%) of 102 intracranial dural AVFs. Again, an association between nonhemorrhagic neurological deficit and Borden type was found in one (2%) of 55 Type I, five (28%) of 18 Type II, and nine (31%) of 29 Type III patients ($\chi^2 = 15.899$, $df = 2$, $p < 0.0001$). When considered in combination, the relationship between aggressive presentation and Borden type was striking: one (2%) of 55 Type I, seven (39%) of 18 Type II, and 23 (79%) of 29 Type III intracranial dural AVFs ($\chi^2 = 54.644$, $df = 2$, $p < 0.0001$). These results are summarized in Table 3.

One patient had a Borden Type I intracranial dural AVF that presented in an aggressive manner. This was a child with a Cognard Type IIA transverse sinus intracranial dural AVF who presented with head enlargement and

retrograde DVS flow (red blood filling the TS). In addition, there is retrograde leptomeningeal venous drainage (red leptomeningeal vein). Selective right occipital artery (*small straight arrow*) angiogram, lateral projection (H), showing a TS nidus that drains anterogradely through the transverse/sigmoid sinuses (*arrowheads*). The intracranial dural AVF also has retrograde DVS flow into the superior sagittal (*curved black arrow*) and straight (*open arrow*) sinuses. The retrograde leptomeningeal venous drainage into the deep venous system is seen (*large straight arrow*). I and J: Borden Type III/Cognard Type III intracranial dural AVF showing retrograde leptomeningeal venous drainage only. Artist's rendering (I) depicting nidus outflow that uses leptomeningeal veins exclusively (*red*). The TS (*blue*) may be occluded or works in isolation carrying venous blood. Selective left occipital artery (*arrow*) angiogram, lateral projection (J) showing a TS (*arrowhead*) intracranial dural AVF. Outflow occurs via leptomeningeal veins such as the vein of Labbé (*curved arrow*). The segment of TS is isolated (*open arrows*). K and L: Borden Type III/Cognard Type IV intracranial dural AVF exhibiting retrograde leptomeningeal venous drainage (with ectasia) only. Artist's rendering (K) similar to that shown in I, but illustrating an ectatic leptomeningeal vein. Right external carotid artery angiogram, lateral projection (L) in which a large posterior middle meningeal branch (*small arrow*) is seen to supply a TS nidus (*large arrow*) that empties exclusively into the vein of Labbé (*curved arrow*). Blood ultimately escapes via other cortical veins, including an ectatic sylvian vein (*arrowhead*) on the way to the cavernous sinus. M: Borden Type III/Cognard Type V intracranial dural AVF, shown in selective right ascending pharyngeal artery (*arrowhead*) angiogram, lateral projection, displaying retrograde leptomeningeal venous drainage with involvement of perimedullary spinal veins. This foramen magnum intracranial dural AVF (*thin arrow*) has retrograde leptomeningeal venous drainage into a dorsal perimedullary spinal vein (*large arrow*).

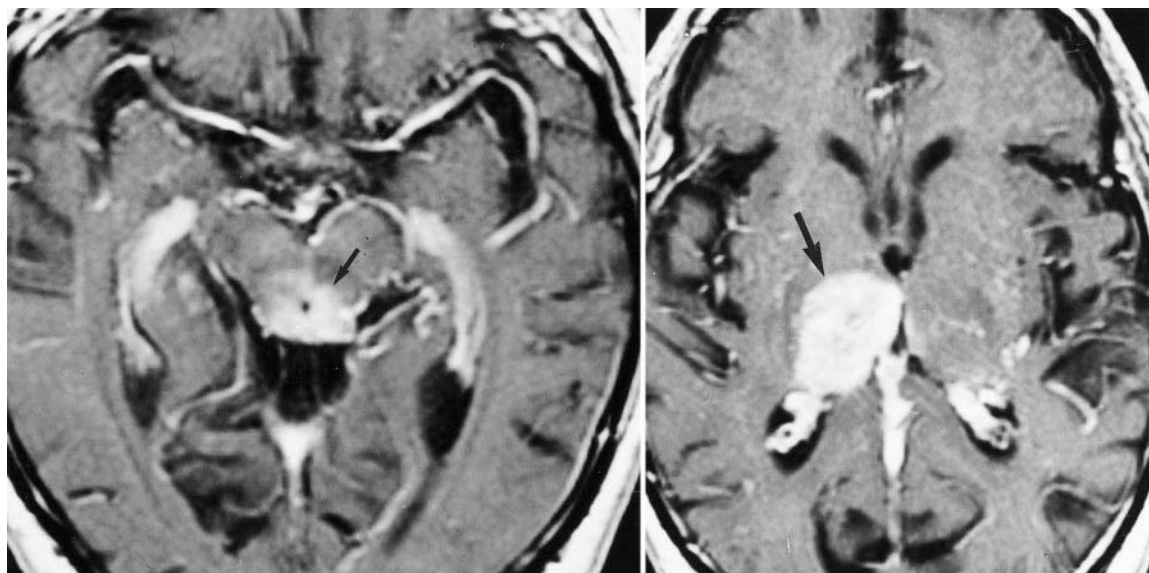


FIG. 2. Contrast-enhanced T₁-weighted axial magnetic resonance images demonstrating contrast enhancement in the territory of leptomeningeal veins in the deep system subjected to retrograde flow from an intracranial dural arteriovenous fistula. *Left:* Dorsal midbrain (*arrow*). *Right:* Right thalamus (*arrow*).

developmental delay. Communicating hydrocephalus was confirmed by MR imaging.

To control for bias toward higher grade lesions in some locations, all Borden Type III patients were assessed to see if lesion location still influenced aggressive presentation. Intracranial hemorrhage or nonhemorrhagic neurological deficit was seen in three-fourths (75%) of the lesions located in the anterior cranial fossa, three (100%) of three located in the foramen magnum, one (100%) of one in the superior sagittal sinus, eight (80%) of 10 in the tentorium, and eight (89%) of nine in the transverse sinus lesions (*p* value was not significant).

Discussion

Borden Type and Aggressive Presentation

Our data show a highly significant correlation between Borden type and either ICH or nonhemorrhagic neurological deficit (*p* < 0.0001). When this classification was proposed by Borden, *et al.*,⁴ they suggested that the logical stratification of venous drainage seen on angiography should be reflected in the relative frequency of aggressive presenting behavior. Although the Cognard classification more than adequately describes the important features of any given intracranial dural AVF, the Borden classification is more compact: Borden Type I lesions, which drain via normal dural channels, pose a minimal threat; Type II lesions, which pose a greater threat, have parasitized leptomeningeal venous conduits but retain dural venous sinus outflow that may be restricted or may require a larger arteriovenous shunt; and Type III lesions, which clearly exhibit the most dangerous course, have a nidus with no dural venous sinus access and drain entirely into leptomeningeal veins. The advantage of the Borden classification lies in its simplicity without loss of predictability.

Cognard Type and Aggressive Presentation

We have been able to reconfirm the utility of the grading system of Cognard, *et al.*,⁶ in accurately predicting aggressive behavior at presentation in terms of either ICH or nonhemorrhagic neurological deficit (*p* < 0.0001). Djindjian and colleagues⁷ recognized the key role of cortical venous drainage in the behavior of intracranial dural AVFs, already demonstrated in earlier series,^{5,9} by establishing a means of classifying intracranial dural AVFs based primarily on their venous anatomy. Cognard, *et al.*, have subsequently restructured this scale, with a detailed stratification that allows for both the directional flow characteristics of a patent dural venous sinus and the acquisition of retrograde leptomeningeal venous drainage. They were able to show good correlation between type and aggressive presentation together with adverse neurological events in their 205 cases. As distinct from our experience, they found that eight of their 17 Type IIA patients presented with signs of raised intracranial pressure. Based on previous theories, they suggest that reduction in cerebrospinal fluid absorption secondary to dural venous sinus and cortical venous hypertension were the likely cause.^{6,12,17}

Retrograde Leptomeningeal Venous Drainage

Although lengthy, the term "retrograde leptomeningeal venous drainage" most accurately describes the single most important feature with which we can recognize or exclude any intracranial dural AVF. The term "cortical venous drainage," although used by most authors in the past,^{3,5-9,11,14} unfortunately leads to the inaccurate assumption that only recruitment of veins normally draining the cortex of the cerebral hemispheres or cerebellum is important. Cortical venous drainage was not seen in many of our patients, who nevertheless had an ICH or nonhemorrhagic

ic neurological deficit that was appropriate to the territory of leptomeningeal veins draining noncortical structures. This was most often seen in the diencephalon with retrograde leptomeningeal venous drainage involving the deep system only (Fig. 2). Borden, *et al.*,⁴ in using the word “subarachnoid,” although failing to describe the leptomeningeal zone, meant to stress the equal importance of veins draining the noncortical central nervous system parenchyma, such as the diencephalon, brainstem, and so forth. Awad and colleagues² seem to be the first to use the term “retrograde leptomeningeal venous drainage” in their literature review and metaanalysis of 360 cases. Involvement of veins traversing the leptomeningeal space that normally drain the brain (or spinal cord), not the dura, is the fundamental concept to be grasped. Anterograde flow in these vessels is normally from the brain (or spinal cord) to a dural venous sinus. When the nidus of an intracranial dural AVF uses a leptomeningeal vein as an outflow conduit (Borden Types II and III), the flow is reversed (toward the brain). This leads to venous hypertension and ischemia, which are often evidenced by the radiological picture of venous congestive encephalopathy (venous territory appropriate to T₂-weighted MR imaging signal hyperintensity) described by Willinsky, *et al.*¹⁸ In our experience, the words “retrograde leptomeningeal” convey the clearest message.

Misconceptions About Intracranial Dural AVFs

Lesion location has attracted undeserved recognition as a sinister characteristic of intracranial dural AVFs. As early as 1973, Aminoff¹ separated these lesions, because of their anatomical location, into an anteroinferior and posterosuperior group, the latter exhibiting a more dangerous course. Since then, the tendency for intracranial dural AVFs in some locations to present more frequently with ICH and nonhemorrhagic neurological deficit has been well established in the literature both in a metaanalysis and in the recent large series reported by Cognard, *et al.*^{2,6} Previously reported frequencies of aggressive presentation for tentorial (92%–97%), anterior cranial fossa (68%–88%), and superior sagittal sinus (50%–65%) lesions are consistent with our findings of 79%, 75%, and 50%, respectively.^{2,6} There are two possible explanations. Simplistically, one could argue that intracranial dural AVFs in some locations do not herald their existence with benign symptoms such as ophthalmoplegia and, therefore, are not discovered until ICH or nonhemorrhagic neurological deficit occurs. However, our data would support the contention of Malik and colleagues,¹⁴ who believed that the poor prognosis of lesions in “dangerous” locations is purely a function of their possession of a more dangerous venous anatomy: that is, regional venous anatomy enhances the possibility of (as in the case of tentorial lesions) or mandates (as in the case of anterior cranial fossa lesions) retrograde leptomeningeal venous drainage in some locations. When we controlled for lesion location by selecting all Borden Type III patients, we found no statistical difference between the locations with regard to frequency of aggressive presentation. This also holds true when comparing all Cognard Types III through V.⁶ Awad and colleagues² state that no location of intracranial dural AVF is immune from aggressive neurological behavior.

Because an intracranial dural AVF in any location can possess retrograde leptomeningeal venous drainage, we would also support this view. We therefore propose that lesion location should only serve to raise the index of suspicion concerning retrograde leptomeningeal venous drainage in certain regions.

The efficacy of the Borden⁴ and Cognard⁶ classification schemes suggests that the venous anatomy of an intracranial dural AVF is the major determinant of the mode of clinical presentation. We must emphasize that our results cannot be extrapolated to predict lesion behavior after presentation. This continues to pose a difficulty, because although much is known and still being published about the natural history of brain AVMs, intracranial aneurysms, and cavernous malformations, surprisingly, the natural history of intracranial dural AVFs has not been addressed. The answer cannot be derived from the available literature, which consists of case reports, small personal series, and location-specific studies. Awad and colleagues,² in their metaanalysis of cases prior to 1990, only provide some theoretical statements in their discussion. Despite a significant follow-up period, the series of 43 intracranial dural AVFs studied by Femand, *et al.*,⁸ deals with transverse sinus lesions and only seven avoided treatment. Besides our own, the only large heterogeneous single-institution study was published in the radiology literature by Cognard, *et al.*⁶ Unfortunately, although 205 cases were followed for a mean of 52 months, the authors’ evaluation did not differentiate events occurring prior to, from those occurring after, presentation in sufficient detail. This lack of knowledge about intracranial dural AVF behavior is of concern because we have observed retrograde leptomeningeal venous drainage in all lesion locations. Some of these intracranial dural AVFs present in a benign fashion (for example, seven cavernous sinus lesions in our series); however, there is insufficient data to predict accurately their future course. Intuitively, the identification of retrograde leptomeningeal venous drainage should be a key issue in determining the natural history of these lesions. We have shown a highly significant correlation between lesion presentation and lesion grade based on venous anatomy. If these classification schemes could be applied efficaciously to predict adverse lesion behavior after diagnosis, they would serve as important tools in planning rational management strategies. We are studying our cohort prospectively with plans to address these important issues.

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